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Frequent Estimation of Arterial End-Tidal Co2 Difference during End-Tidal CO2-Management in Patients with Elevated Intracranial Pressure

Stanlies D'Souza

Baystate Health, dsouzastan@yahoo.com

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Introduction

Our goal is to highlight the importance of frequently estimating PaCO₂ to identify the degree of dead space ventilation and target end-tidal CO₂ in patients with acutely elevated intracranial pressure (ICP) presenting for intracranial surgery.

Case Description

A 72-year-old female with deteriorated neurological status and significantly elevated ICP due to a massive postoperative intracranial hematoma, presented for evacuation of the hematoma. Patient’s initial arterial end-tidal CO₂ gradient was 14mmHg (PaCO₂ 42 mmHg and end-tidal CO₂ 28 mmHg). Based on this, patient was further hyperventilated to an end-tidal CO₂ of 25 mmHg.

Discussion

In cases of intracranial bleeding, a massive systemic catecholamine surge causes intense peripheral and pulmonary vasoconstriction. This in turn increases dead space ventilation and higher arterial end- tidal CO₂ gradient. Normocarbica decreases cerebral blood flow while hypercarbia further elevates ICP, with catastrophic consequences.

PaCO2 management in a patient with elevated ICP

- Maintain normocarbica (PaCO2 35-40 mm Hg)
- Hypercarbia abolishes cerebral autoregulation, decreasing blood flow via cerebral vasoconstriction while increasing it via vasodilation

Pathophysiology

Following acute intracranial events (such as an intracranial bleed, acute ischemic stroke (AIS) or a traumatic brain injury (TBI)), massive sympathetic discharges of catecholamine and cytokines occur.¹ This acute inflammatory response causes systemic-end organ changes and, in turn, has **cardiac** and **pulmonary** effects. The degree to which cardiac and pulmonary systems are involved depends on the severity of the intracranial event.

Cardiac Changes²

- EKG: ST and T wave changes, sinus tachycardia and tachyarrhythmias
- Wall motion abnormalities show on transesophageal echocardiography
- Changes are due to catecholamine-induced cardiomyopathy, a condition known as “stress-induced cardiomyopathy” or “Takotsubo cardiomyopathy”
- In severe cases, cardiogenic pulmonary edema occurs.
- The degree of cardiac involvement correlates to the severity of the intracranial event.

Pulmonary Changes³

- Pulmonary involvement is due to cytokines released following an intracranial event
- Cytokines increase pulmonary capillary permeability and fluid leak.
- These changes can result in pulmonary edema in severe cases
- The degree of pulmonary involvement correlates to the severity of the intracranial event.

Intracranial bleeding and its effect on pulmonary ventilation

- ✓ Acute intracranial bleed elicits systemic inflammatory response.
- ✓ Massive amounts of catecholamine surge cause pulmonary vasoconstriction.
- ✓ Pulmonary vasoconstriction decreases blood flow to the alveoli.
- ✓ Decreased alveolar blood flow in the presence of normal ventilation results in dead space ventilation.
- ✓ Increased dead space ventilation increases PaCO2 and end-tidal CO2 gradient.
- ✓ The degree of dead space ventilation depends on the severity of the intracranial event.

Why is frequent estimating of PaCO2 necessary?

- With an acute intracranial bleed in a closed cranium, catecholamine surge is high, resulting in greater dead space ventilation.
- The catecholamine surge is likely to decrease following the opening of the dura. This will reduce dead space ventilation and lower the previously-elevated PaCO2 end-tidal CO2 gradient.

CONCLUSION

Since PaCO2 has a profound effect on cerebral vasculature, accurately managing it and its effect on ventilation is essential during craniotomy for an intracranial hemorrhage. As described above, frequently varying dead space ventilation causes changes in PaCO2 end- tidal CO2 gradient. Therefore, we emphasize frequent PaCO2 measurements and regular estimating of dead space ventilation gradient during anesthetic management of a craniotomy for intracranial hemorrhage.